POSTOPERATIVE GAS BACILLUS INFECTION OF THE ABDOMINAL WALL

S

BUTLER

POSTOPERATIVE GAS BACILLUS INFECTION OF THE ABDOMINAL WALL*

By Dell D. Butler, B.S., M.D.,
Fellow in Surgery, The Robert Packer Hospital (Guthrie Clinic)
Sayre, Pa.

Gas bacillus infection of the abdominal wall is a very rare complication following laparotomies. In spite of abundant literature on the fecal anaërobes and infections caused by them, extensive search has revealed only a few references to this particular condition. Its serious import when it does occur, and the appearance of two cases at the Robert Packer Hospital, within two years, have prompted a study to determine what risk of such infection besets the average patient.

In this discussion gas bacillus infection includes all infections caused by fecal anaërobes, such as Bacillus tetani, B. Welchii, Vibrian septique, B. œdematiens, B. fallox, B. sporogenes, B. histolyticus, B. putrificus, etc. Those mentioned are the most frequently encountered anaërobic bacilli and clinically the most important. Of this group B. Welchii is undoubtedly of prime importance.

The Robert Packer Hospital is situated in an agricultural district. Within a radius of 100 miles there are only thirteen cities of 25,000 or more population. One-half or more of its patients are farmers or are engaged in outdoor rural occupations. The frequency of anaërobes in such an environment should be greater than in the metropolitan districts. This presumption is strengthened by the fact that during the period covered by this report there occurred, in addition to the cases already mentioned, five cases of tetanus and three cases of

*Short thesis submitted for M.S. degree to the Post-Graduate School of the University of Pennsylvania.

gas bacillus infection of other parts as follows: knee, arm and leg. However, during the past fifteen years of this hospital's present management, postoperative gas bacillus infection has occurred only twice in approximately 7000 laparotomies.

In view of the known habitat of the fecal anaërobes, a study of the intestinal flora of this particular district was undertaken, even though similar studies had been made by other observers in other districts. A review of the other reports reveals many discrepancies.

Dungeon and Sargent[1] in a study of the bacteriology on appendices in England in 1905, found no anaërobes. On the other hand, Lanz and Tavel[2] found bacillus oedematis maligni in 49 out of 139 cases of appendicitis. Under bacillus edematis maligni they included various other anaërobes, including bacillus Welchii. Runeberg[3] in 1908 found bacillus Welchii once in 14 cases of appendicitis. Hyde and Frederick's Clinic in Marburg[4], in 1911, concluded that anaërobic bacteria were found in 100 out of 102 cases of appendicitis studied. Grigoroff[5] found bacillus Welchii present nine times in 31 cases of appendicitis. Sinomds[6] and Jennings[7] had similar results. Welch[8], Flexner[9], Wright[10], Stokes[11] and others were able to isolate bacillus Welchii from 22 per cent. of peritoneal exudates following peritonitis.

Winter[12] in 1889 was the first to describe emphysema of the abdominal wall after laparotomies, and reported two cases. Madalener[13] reported two more cases, and claimed that the posture with the hips elevated was of predisposing moment in the origin of the emphysema, while Leopold and Brosin[14] had already proved that the condition could arise after operations, regardless of the patient's position. Heil[15] contended that emphysema could only develop after imperfect closure of the incision. He collected 20 cases from the literature.

5

Russell[16] reported two cases in 1897 from the gynecological service of Johns Hopkins Hospital, one following suspension of the uterus for retroflexion, the other following a panhysterectomy in which the intestine was inadvertently opened.

Among the cases collected by Drs. Welch and Flexner[17] are several following perforation of the gut, showing that the organisms must exist in the intestine. This is of particular interest when one considers the frequency of intestinal injuries in abdominal operations. None of the above cases were bacteriologically proved.

In very recent literature two cases have been reported of true gas bacillus infection of the abdominal wall. Bier[18] reported a case which developed in forty-eight hours after appendectomy. Under appropriate treatment, instituted early, the patient made a good recovery.

Ochsner and Schmidt[19] reported a case developing after an appendectomy for a perforated appendix associated with abscess formation. Likewise this patient made a good recovery.

An extraordinary case has been reported by Eaton[20], of gas bacillus infection involving the neck, supra- and infra-clavicular spaces, following a perforated gastric ulcer. Rapid involvement of the entire body took place within an hour after death. Necropsy revealed the presence of gas bubbles in the stomach wall, suggesting that here was the seat of primary infection, particularly as no gas bubbles were observed in the intestinal mucosa.

One case from the Robert Packer Hospital followed an appendectomy; the other followed a colostomy. The histories are as follows:

Case I.— Mrs. H.H., aged 20, stenographer, admitted to this clinic March 3, 1904, complaining of pain and tenderness in the right lower abdomen, associated with nausea, vomiting and slight diarrhea. Examination revealed intense soreness and rigidity in the right lower abdomen. Vaginal examination

showed a small mass in the region of the right tube and ovary. The laboratory findings were: W.B.C. 21,000, polymorphonuclears 92%, lymphocytes 8%. Urine showed a cloud of albumin and many granular casts. T.P.R. 98.4 - 100 - 24. A diagnosis of acute perforated appendicitis was made. Immediate appendectomy was performed through a McBurney muscle-splitting incision. An acutely inflamed gangrenous perforated appendix was found lying behind the cæcum. A small abscess had formed. A right salpingitis known to be of long standing was also found. The abdomen was drained by two rubber drains.

The patient seemed listless after operation, but aside from that there were no alarming symptoms. The temperature and pulse were as follows: 101 F - 100. Seventy-two hours after operation the skin of the lower right quadrant of the abdomen appeared bronzed and ædematous and was crepitant upon palpation. The pulse rate rose rapidly to 134 per minute. These findings aroused the suspicion of a gas bacillus infection of the abdominal wall; hence immediate multiple incisions through the skin and subcutaneous tissues were made and a watery brownish, foul-smelling pus evacuated. There was no evidence that the deeper structures of the abdominal wall were involved by this infection. Anaërobic cultures were positive for bacillus aerogeneous capsulatus. The wounds were dressed every four hours with normal saline solution. One hundred and twenty hours later additional multiple incisions were made because the infection had continued to progress. After that the patient gradually improved and was discharged as cured forty-nine days after the onset of the infection.

Case II.- J.R.F., aged 33, an American, insurance agent, entered this clinic June 3, 1925, complaining of severe pain in the right lumbar region and the right lower abdomen. This attack had begun suddenly one day before admission. The pain had not been referred to any other parts, nor had it been associated with any nausea, vomiting or urinary symptoms. A similar attack one week previous to admission had subsided in three days. Attacks of pain in the right side dated back fifteen years. There had been no previous operations. Physical examination was negative except for slight distention, rigidity over the entire right abdomen and very active peristalsis. The admission findings were: T.P.R. 99 - 74 - 20. W.B.C. 9,500, polymorphonuclears 81%, small mononclears 19%. Urine showed a few pus cells. Roentgenogram of the kidneys and ureters was negative. A provisional diagnosis of acute intestinal obstruction of unknown origin was made.

Immediate laparotomy through a right rectus incision was performed. An acute intestinal obstruction due to volvulus of the cæcum, ascending colon and part of the transverse colon was found. The volvulus was caused by adhesions between the ascending and transverse colon. These adhesions were separated,

relieving the obstruction. Over-distention of the large bowel was relieved by a caecostomy fashoned after a Witzel enterostomy. Sixty-seven hours after operation the patient developed a gas bacillus infection of all the structures of the abdominal wall. The pulse rate at this time was 180 per minute. Immediate multiple incisions were made through the skin, subcutaneous tissues, fascia and underlying muscles. The wounds were dressed by the Carrel-Dakin technic. Patient died ninety-six hours after operation in deep toxemia.

Note that in the first case the infection was limited to the superficial tissues only, while in the second case all the structures of the abdominal wall were involved.

Prompted by these two cases the following studies of intestinal flora were undertaken:-

1. Bacteriological studies of appendices removed at operation.
2. Bacteriological studies of appendiceal abscesses.
3. Bacteriological studies of large gut and ileum.
4. Bacteriological studies of free pus in the peritoneal cavity.

Cultures were taken under aseptic conditions and planted in litmus milk. The resulting cultures were incubated continuously, the reactions being noted at twelve hour intervals. After seventy-two hours all cultures were examined microscopically. Cultures showing early stormy fermentation or rapid clotting of the milk, associated with the production of gas, were examined immediately for the presence of bacillus Welchii or other anaërobes. In all sixty-nine cultures were examined at varying periods after original cultures. These were distributed as follows:-

From appendiceal abscesses,	5.
From lumen of excised appendix,	60.
From lumen of large gut,	2.
From free pus in peritoneal cavity,	1.
From lumen of excised ileum,	1.

Of the five cultures of appendiceal abscesses four showed formation of acid and gas, two within the twelve hour period, while one culture

showed no change whatever.

Thirty-seven cultures from excised appendices showed the production of acid and gas associated with clot formation, one within twelve hours of original culture. Seventeen cultures showed only the production of acid, while in six cultures there was no change.

There was acid and gas production in all three cultures from the gut.

In the culture from pus in the peritoneal cavity, acid, but no gas, was produced.

When examined microscopically none of these cultures showed the presence of bacillus Welchii or other anaërobes, nor did any of the patients from whom these cultures were taken develop gas bacillus infection of any part. Nearly all positive cultures showed bacillus coli, diphtheroids or streptococci in varying combinations.

CONCLUSIONS.

I. Postoperative gas bacillus infection of the abdominal wall is a very rare complication, but serious when it occurs.

2. There is relatively little risk as gas producing anaërobes were not found in any cultures made.

3. Suspicion of such a complication should be aroused by a sudden increase in pulse rate, with or without a rise in temperature, in a patient who is not doing well after operation.

4. The diagnostic signs are copper colored bronzing of the skin, with edema, brownish, foul-smelling discharge from the wound, crepitation, and positive bacteriological smears.

5. Treatment should be prompt multiple incisions and free air image.

6. The ~~diagnosis~~ prognosis depends upon the extent of the infection.

Bibliography

1. Dungeon and Sargent. Quoted by Jennings, J.E.: The Rôle of Bacillus Welchii in Gangrenous Appendicitis, New York Med. Jour., CXVII, 682-685, June 6, 1923.

2. Lanz and Tovel: Quoted by Jennings.

3. Runeberg: Quoted by Jennings.

4. Hyde and Frederick: Quoted by Jennings.

5. Grigoroff: Quoted by Jennings.

6. Simonds: Quoted by Jennings.

7. Jennings, J.E.: The Rôle of Bacillus Welchii in Gangrenous Appendicitis, New York Med. Jour., CXVII, 682-685, June 6, 1923.

8. Welch: Quoted by Jennings.

9. Flexner: Quoted by Jennings.

10. Wright: Quoted by Jennings.

11. Stokes: Quoted by Jennings.

12. Winter: Quoted by Russell, W.W.: Postoperative Emphysema of the Abdominal Wall, Amer. Jour. of Obst., XXXV, 517, April, 1897.

13. Kamalener: Quoted by Russell.

14. Leopold and Brosin: Quoted by Russell.

15. Heil: Quoted by Russell.

16. Russell, W.W.: Postoperative Emphysema of the Abdominal Wall, Amer. Jour. of Obst., XXXV, 517, April, 1897.

17. Welch and Flexner: Quoted by Russell.

18. Bier, Edwin: Ann. Surg., LXXXI, 1177, 1925.

19. Ochsner, A.J., and Schmidt, C.H.: Gas Bacillus Infection Originating in Gangrenous Appendix, S. Clinics N.A., V, 911-913, August, 1925.

20. Boyton, W.A.: Gas Bacillus Infection with Perforated Gastric Ulcer, Boston Med. and Surg. Jour., CXCIII, 507-509, September 10, 1925.

RESULTS OF BACTERIOLOGICAL INVESTIGATIONS

PATIENT	DATE OF ADMISSION	POSTOPERATIVE DIAGNOSIS	SOURCE OF CULTURE	LITMUS MILK CULTURE					MICROSCOPIC SMEARS OF CULTURES
				Neg.	Acid & Gas 12H.	Acid & Gas 24H.	Acid & Gas 72H.	Acid & no Gas	
M.H.	8-27-25	Appendicitis, chr.	*L.A.	-					Negative
M.S.	8-31-25	Appendicitis, acute Hematogenous cyst ovary, right	L.A.	-					Negative
L.C.S.	9- 1-25	Appendicitis, acute gangrenous, ruptured Subphrenic abscess	L.A.	-					Negative
R.Y.	9- 6-25	Appendicitis, acute gangrenous, ruptured	Free pus peritoneal cavity					+	Negative
G.M.	9- 7-25	Appendicitis, chr. recurrent	L.A.					+	Negative
L.P.	9- 9-25	Abscess, appendiceal	*A			+			Anerobes -neg. B. coli
M.B.	9-11-25	Abscess, appendiceal	A	-					Negative
A.S.	9- 7-25	Appendicitis, sub-acute	L.A.					+	Negative
M.H.	9-12-25	Appendicitis, acute	L.A.					+	Negative
J.B.	9-16-25	Appendicitis, acute gangrenous, perforated	L.A.					+	Negative
R.A.	9-13-25	Appendicitis, acute	L.A.					+	Negative
H.A.	9-16-25	Typhoid fever Appendicitis, acute	L.A					+	Negative
C.C.	9-30-25	Appendicitis, acute	L.A.					+	Negative
C.C.	9- 4-25	Adenocarcinoma cecum	Lumen cecum		+				B. coli
G.A.	9-20-25	APPENndicitis, acute gangrenous	L.A.					+	B. coli
H.K.	9-25-25	Fibroid uterus Appendicitis, chr.	L.A.				+		B. coli
H.J.	9-25-25	Appendicitis, acute	L.A.					+	B. coli
B.K.	9-26-25	Appendicitis, acute	L.A.				+		B. coli
T.B.	9-25-25	APPENdicitis, sub-acute	L.A.					+	B. coli Diphtheroids
A.L.	9-25-25	Appendicitis, chro.	L.A.					+	Anerobes -neg.
A.W.	9-27-25	Appendicitis, acute	L.A.					+	B. coli
J.L.	9-29-25	Appendicitis, acute gangrenous	L.A.					+	B. coli
E.C.	9-31-25	Appendicitis, sub-acute	L.A.	-					Negative
B.W.	9-30-25	Appendicitis, sub-acute	L.A.					+	B. coli
W.L.	10- 6-25	Abscess, appendiceal	A		+				B. coli
E.R.	10- 2-25	Appendicitis, acute	L.A.				+		B. coli
E.W.	10- 6-25	Appendicitis, acute	L.A.				+		B. coli
A.M.	10- 8-25	Hernia, umbilical, strangulated with gangrene of ileum	Lumen excised ileum				+		B. coli
F.E.	10-12-25	Cholecystitis, chr. Appendicitis, chr.	L.A.				+		B. coli Staphlococci
H.C.	10-14-25	Cholecystitis, chr. with cholelithiasis Appendicitis, chr.	L.A.				+		B. coli
J.K.S.	10-12-25	Appendicitis, acute	L.A.		+				B. coli
H.P.	10-14-25	Appendicitis, chr.	L.A.				+		Streptococci B. coli
J.M.	10-15-25	Appendicitis, acute gangrenous, perforated	L.A.				+		B. coli
H.F.	10-16-25	Appendicitis, acute	L.A.					+	Negative
S.C.	10-18-25	Appendicitis, acute	L.A.				+		B. coli
H.H.	10-20-25	Appendicitis, chr.	L.A.				+		B. coli Diphtheroids
A.R.	10-20-25	Appendicitis, sub-acute	L.A.					+	B. coli
W.C.	10-22-25	Appendicitis, acute	L.A.				+		B. coli
P.E.	10- 9-25	Cholecystitis, chr. Appendicitis, chr.	L.A.				+		B. coli
V.H.	10-24-25	Appendicitis, acute	L.A.				+		B. coli
H.C.	10-25-25	Appendicitis, chr.	L.A.				+		B. coli
F.O.	10-26-25	Appendicitis, acute gangrenous, perforated	L.A.			+			B. coli
D.R.	10-27-25	Abscess, appendiceal	A		+				B. coli
A.F.	10-31-25	Appendicitis, chr. recurrent	L.A.			+			B. coli
S.C.	11- 9-25	Appendicitis, acute	L.A.				+		B. coli Diphtheroids
G.B.F.	11-10-25	Appendicitis, acute	L.A.				+		B. coli
J.C.	11-10-25	Appendicitis, acute	L.A.					+	B. coli Streptococci
T.L.	11-13-25	Appendicitis, acute	L.A.				+		B. coli Diphtheroids
M.G.	11-16-25	Appendicitis, chr.	L.A.				+		B. coli Diphtheroids
C.L.	11-16-25	Cholecystitis, chr. Appendicitis, chr.	L.A.				+		B. coli Diphtheroids
O.D.	11-18-25	Fibroid uterus Appendicitis, chr.	L.A.				+		B. coli Diphtheroids
B.H.	11-18-25	Appendicitis, sub-acute	L.A.				+		B. coli Diphtheroids
F.T.	11-18-25	Hernia, ventral Appendicitis, sub-acute	L.A.				+		B. coli Diphtheroids
D.O.	11-19-25	Retroversion, uterus Appendicitis, chr.	L.A.	-					Negative
G.H.	11-22-25	Appendicitis, acute gangrenous, perforated	L.A.	-					Negative
J.S.	12- 5-25	Appendicitis, acute	L.A.				+		B. coli Diphtheroids
M.C.	12- 5-25	Appendicitis, acute gangrenous	L.A.				+		B. coli Streptococci Diphtheroids
D.L.	12- 4-25	Appendicitis, chr. recurrent	L.A.				+		B. coli Diphtheroids
R.W.	12- 7-25	Appendicitis, acute	L.A.			+			B. coli Streptococci Diphtheroids
A.W.	12- 6-25	Appendicitis, chr. Cholecystitis, chr. with stones	L.A.				+		Yeast fungus
D.R.	12-13-25	Appendicitis, acute	L.A.			+			B. coli Diphtheroids
R.C.	12-13-25	Hernia, inguinal Appendicitis, acute	L.A.			+			B. coli Streptococci
L.C.	12-16-25	Appendicitis, acute gangrenous	L.A.			+			B. coli Staphlococci
J.F.	12-21-25	Appendicitis, acute Gangrenous, perforated	L.A.				+		B. coli G.pos. cocci
R.F.	12- 7-25	Cholecystitis, chr. Appendicitis, chr.	L.A.				+		B. coli Diphtheroids
R.N.	1-10-26	Appendicitis, acute gangrenous, perforated	L.A.				+		Large G. neg. bac., B.coli, Diphtheroids
M.J.	1-10-26	Appendicitis, chr. recurrent	L.A.				+		G.neg. bac. Diphtheroids
E.L.	1-11-26	Abscess, appendiceal	A				+		Streptococci G.neg. bac.
H.L.	1- 7-26	Carcinoma, transverse colon	Lumen large gut				+		B. coli

*L.A. .... Lumen of excised appendix
*A .... Appendiceal abscess